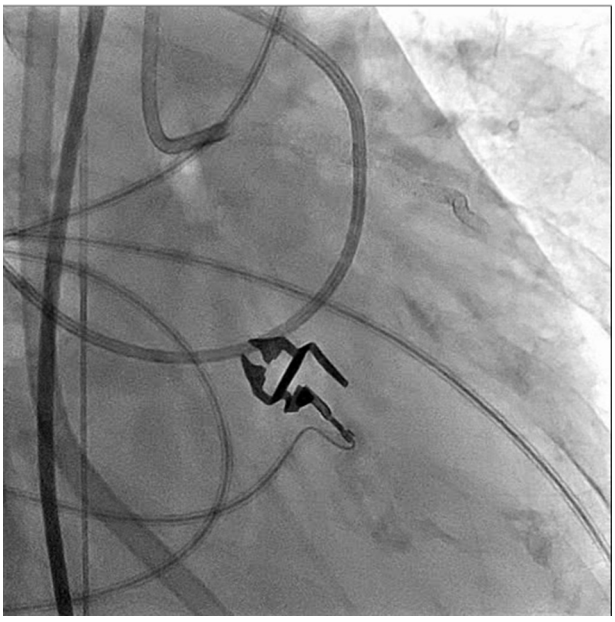
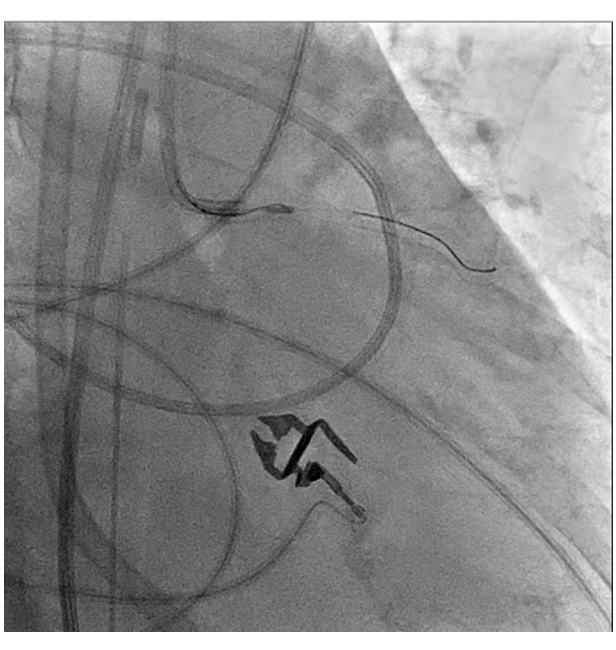
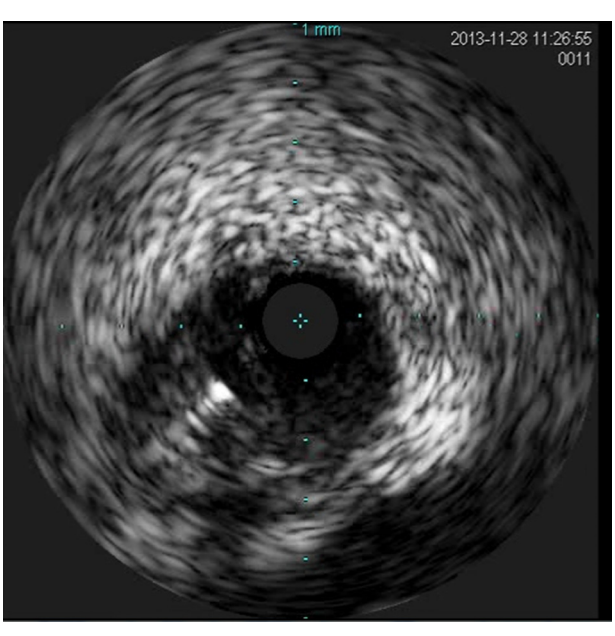


[INTERVENTIONAL MANAGEMENT]
Procedural step. In spite of intensive treatment, her heart failure did not seem to be getting better. So that, we performed 2nd PCI for her occluded LAD.IVUS image showed large dissection at proximal LAD and oppressed small true lumen. Though it was very difficult to insert the guide wire into true lumen, fortunately, we could pass the guide wire with IVUS guidance. After the guide wire crossing, we implanted two drug eluting stents and achieved good recanalization. (System) Approach: Lt. femoral, GC: Launcher 8Fr EBU3.5, GWRunthrough Floppy, Sion Blue, Micro catheterFINECROSS GT, IVUS: Eagle Eye Platinum





Case Summary. We experienced a LAD ostial large dissection caused by PCI procedure, and successfully treated by IVUS guide PCI in 2nd session.If we encounter the coronary ostial large dissection, IVUS guided guide wire control may be helpful for wire crossing to the small true lumen.

TCTAP C-118
Without Proper Collateral Flow, Antegrade Flow Must Be Alive

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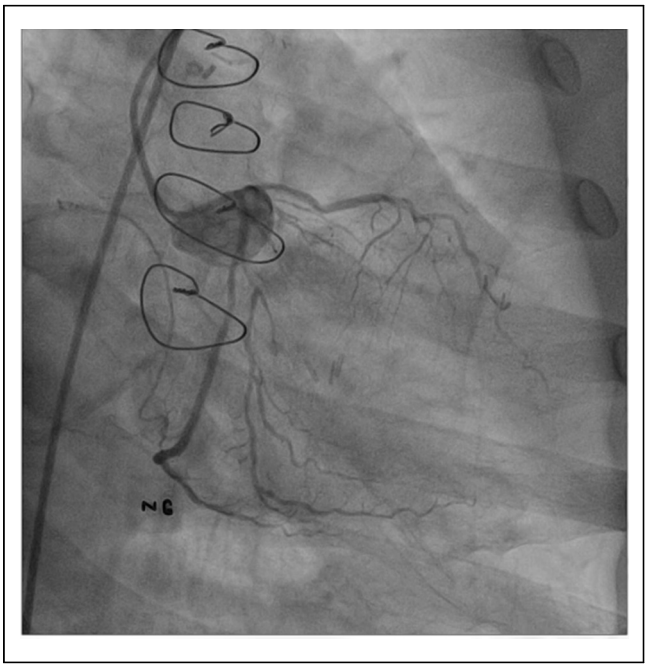
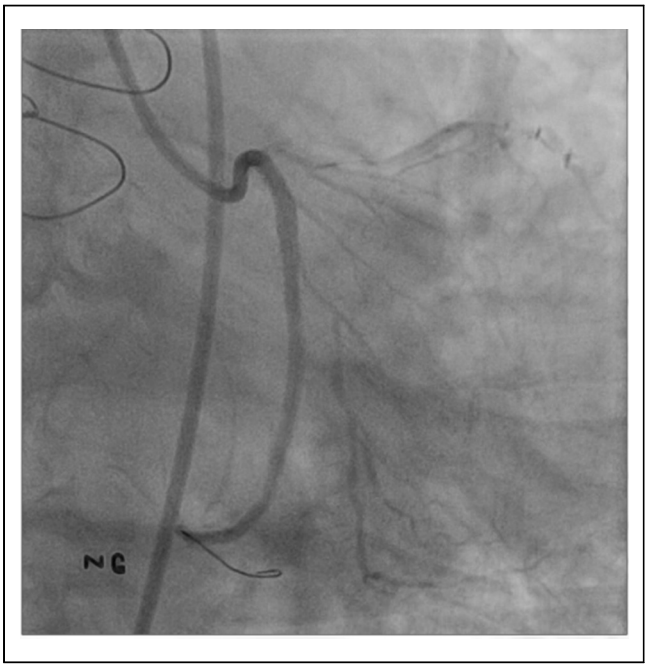
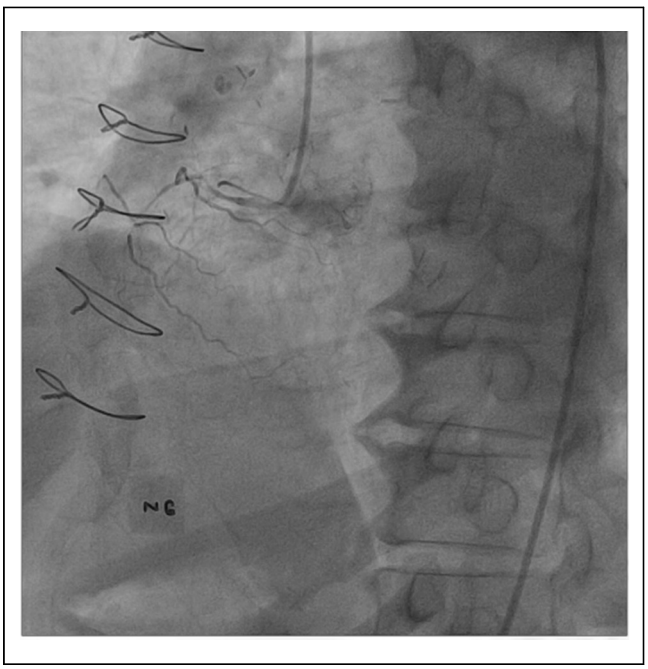
[CLINICAL INFORMATION]
Patient initials or identifier number. DHK

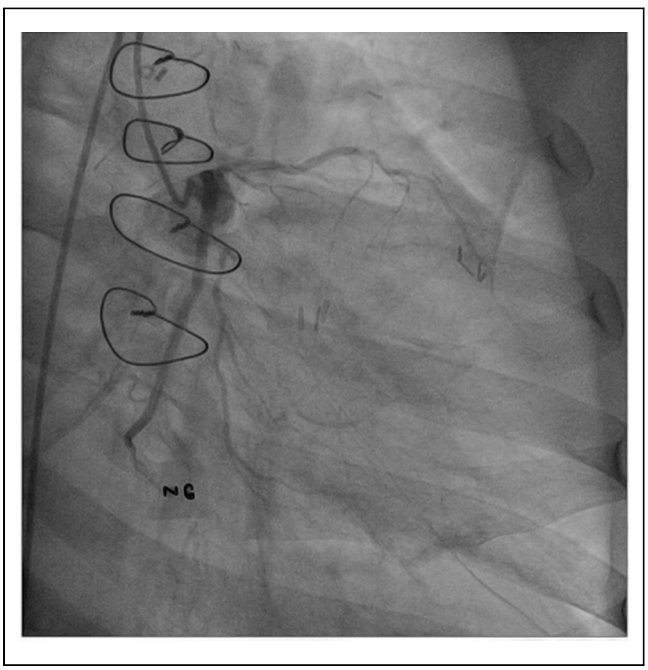
Relevant clinical history and physical exam. A 50-year-old man,known to have diabetes, hypertension, dyslipidemia and previous CABG - LIMA to LAD, radial artery to OM1, SVG to distal PL, had been admitted in October 2013 due to unstable angina. The first CAG showed chronic total occlusion of LAD, RCA and thrombotic 95% stenosis of LCX. LIMA and radial artery grafts were patent, but SVG was invisible. Progression of LCX lesion was suspected the reason of angina and two stents (Xience-V 3.0 * 15 mm, 2.75 * 38 mm) were deployed to p-dLCX.

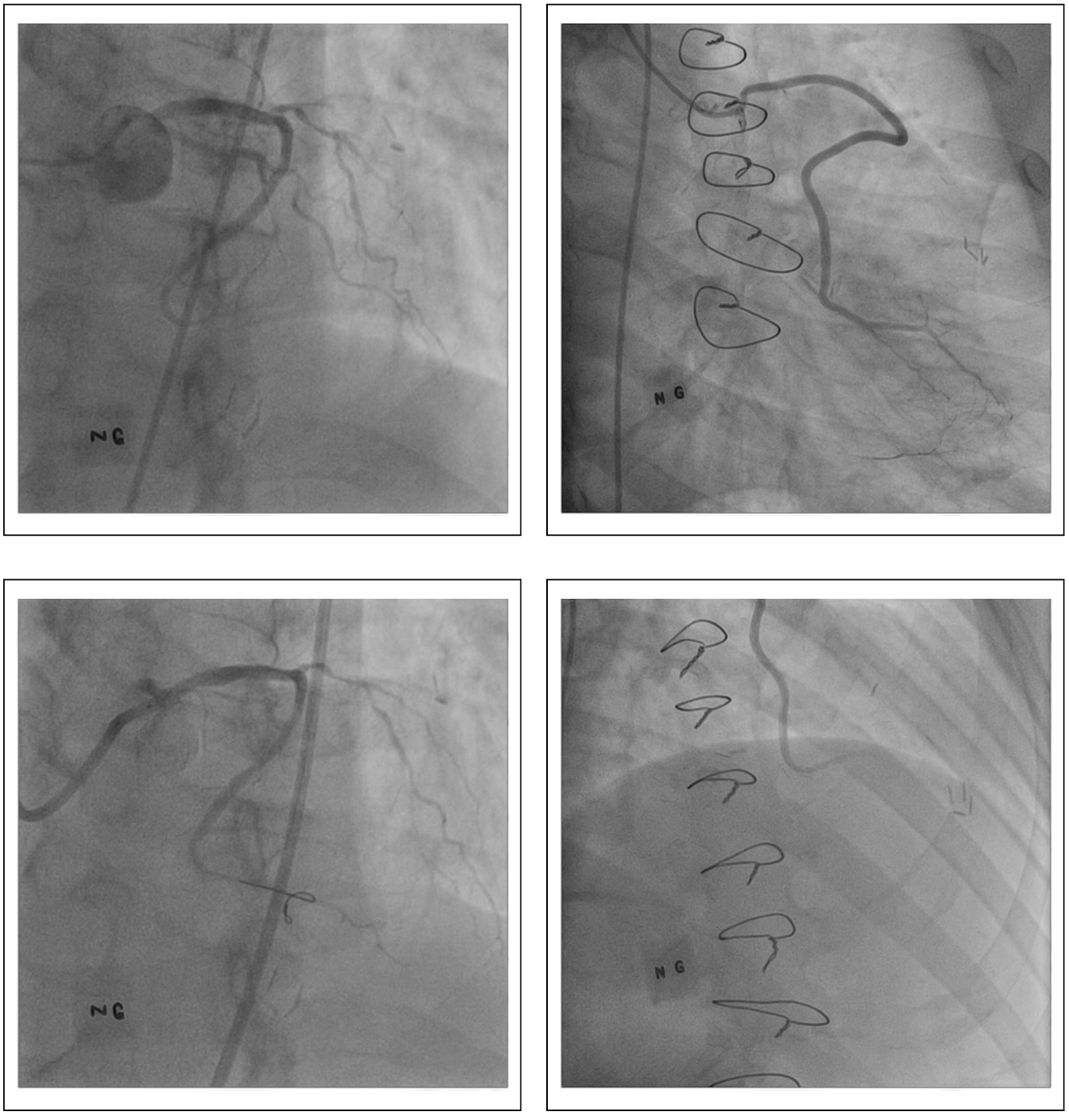




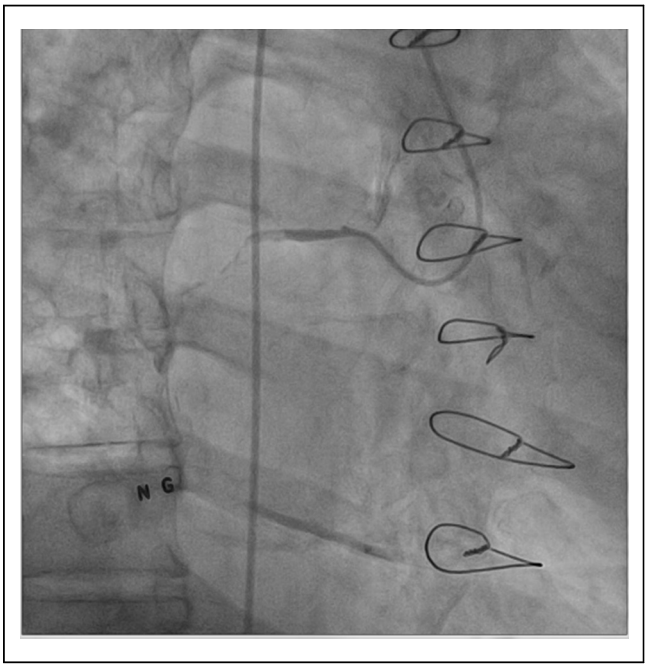
Relevant test results prior to catheterization. Despite the revascularization of the proximal LCX, the patient complained of persistent pain, so another stent (Xience-V 2.5 * 12 mm) was deployed to far distal LCX in November 2013. Nevertheless the patient had continuous angina and was readmitted in September 2014 for reevaluation. ECG and Chest X ray were unremarkable and the echocardiography showed normal LV systolic function (EF= 62 %) and no regional wall motion abnormality.

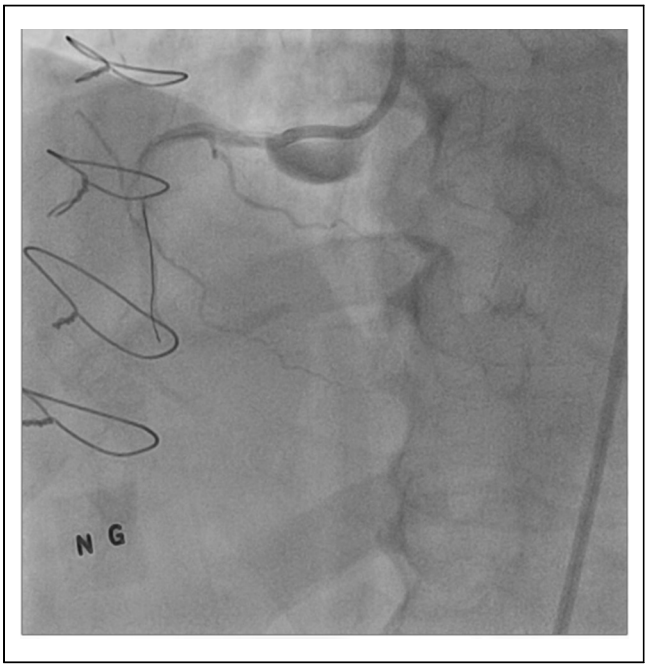






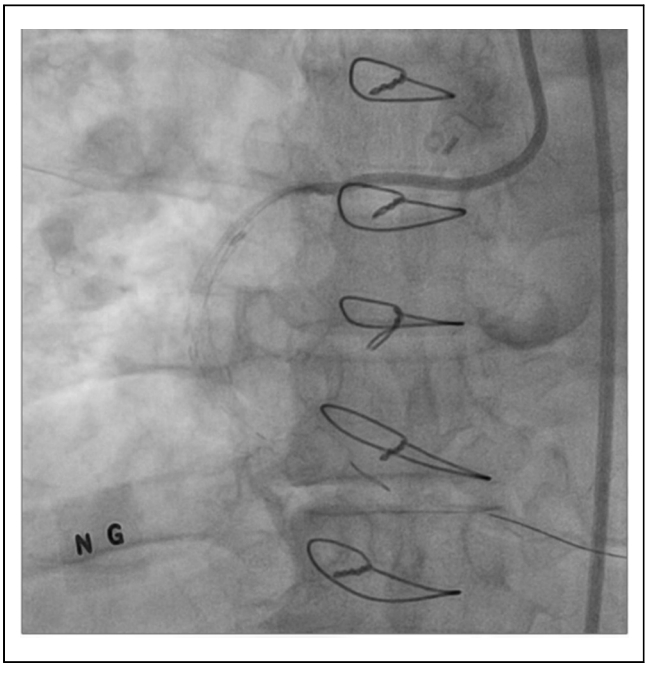
Relevant catheterization findings. The CAG showed near total occlusion of LAD, RCA and patent stents in LCX. The graft angiography showed patent LIMA to LAD and patent radial artery to OM1 grafts. SVG to PL was regarded occluded again as before. The absence of any remarkable collateral flow to RCA territory despite of the chronic total occlusion of RCA lesion made us to realize that distal RCA was being supplied by another feeder unnoticed yet. Most probably SVG might not be totally occluded but slit like patent.

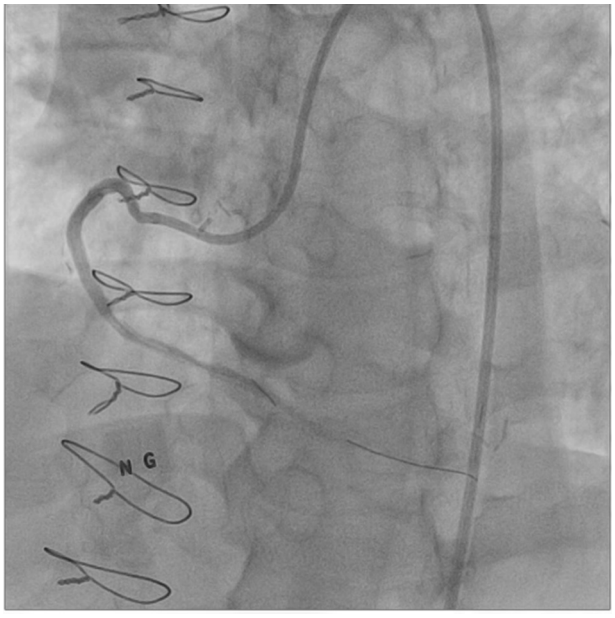


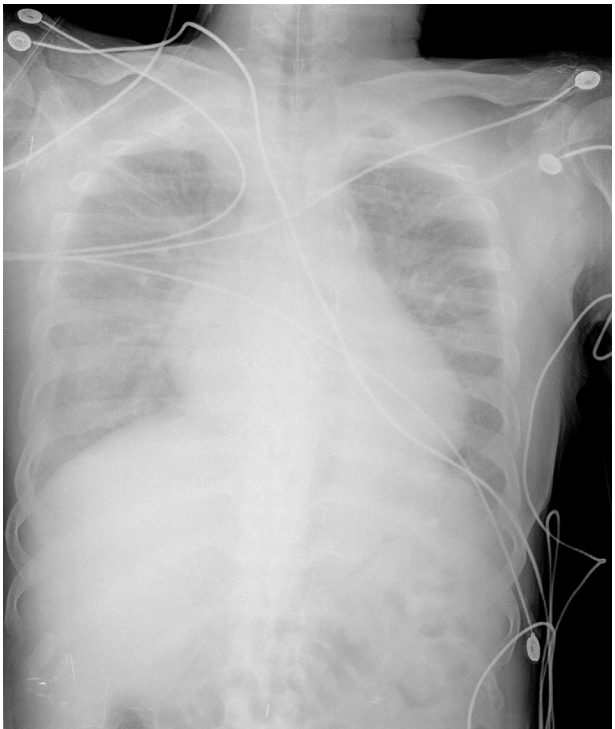


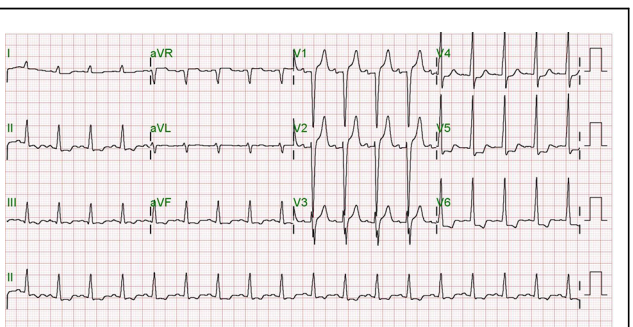
[INTERVENTIONAL MANAGEMENT]

Procedural step. At the end of repeated trial, we could find the SVG from aorta and thrombotic near total occlusion of SVG still supplying PL branch. Revascularization for either RCA or SVG could be considered, but because the outcome of angioplasty of vein grafts is relatively poor or even unknown in drug eluting stent era, we tried PCI for RCA first. Limitation in advancing a wire through the proximal RCA made an ostial dissection and false lumen around aorta. To make it worse, because of the tortuous RCA, the wire hardly passed and made false lumen in mid to distal RCA. Wire passage was tried again with the guidance of IVUS, but minor extravasation appeared in distal RCA, so PCI for RCA was stopped and we converted to the PCI for SVG. Bare metal stent (Prokinetics 3.0 * 22 mm) was deployed to close the RCA ostial intimal dissection and block the extension of dissection up to ascending aorta. A Bare metal stent was chosen as well because it was expected to be occluded earlier with higher probability than a drug eluting stent to prevent flow competition with SVG flow in case SVG revascularization will be undergone. IVUS showed huge amount of organized thrombi in SVG. Thrombectomy was done with a filter wire applied to distal SVG to prevent embolism. Two stents were successfully deployed in SVG to PL (Endeavor 3.0 * 38 mm, 3.0 * 22 mm) and TIMI 3 flow recovered from SVG to distal PL branch. The patient's symptom markedly improved after revascularization and he is doing well at present.









Case Summary. Complex coronary anatomy and multiple lesions make us interpret with difficulty regarding which lesions are culprits in patients presenting worsening angina. Based on this case, we can address that since the poor collateral flow to the chronic ischemic area is a strong clue that antegrade flow via bypass graft is still alive and acutely deteriorated, every effort to visualize all the channel going to ischemic area should be done.

TCTAP C-119

PCPS Supported Successful PCI for Complex Coronary Artery Disease with High Surgical Risk: Poor LVEF, Severe Aortic Valvular Stenosis, Tight Left Main Stenosis with Triple Vessel Disease

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[1]Seoul National University Boramae Medical Center, Korea (Republic of)

[CLINICAL INFORMATION]

Patient initials or identifier number. SSG

Relevant clinical history and physical exam. 60 years old male presented with sudden dyspnea, chest pain and syncope since 1 day ago. He was on hemodialysis since 1999 years, and on medication due to chest pain with suspicious RCA territory infarction on echocardiography (on February 2013), however, coronary angiography could not be done because of gross hematuria.

Risk factors: HTN, DM ESRD on HD, current smoker

Physical exam: blood pressure 96/53 mmHg-heart rate 94 /min, rales on whole lung field

Relevant test results prior to catheterization.

1. Chest X-ray: pulmonary edema
2. ECG: ST depression at V4-6, II, III, aVF
3. Lab: CK 76 U/L,CK-MB 1.7 ng/mL, Troponin-I 0.21 ng/mL, Pro-BNP 32,002 pg/mL, pH 7.445, pCO2 38.7 mmHg, pO2 49.5 mmHg, O2 saturation 83.2 %, HCO3 26.0 mEq/L, BUN/Cr 38/5.89 mg/dL
4. Echocardiography: EF 32 %, global hypokinesia, dilated LV cavity, low pressure gradient severe AS (Vmax 3.06 m/sec, mean pressure gradient 37 mmHg), severe pulmonary hypertension (pulmonary artery pressure 75 mmHg)